



Main etiological factors and comorbid pathology in severe cerebral palsy

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Abstract

Introduction The largest number of factors contributing to the development of cerebral palsy (CP) relate to the pre- and intranatal periods. Premature birth and low birth weight are the most important predictors of cerebral palsy and are associated with persistent brain and motor disorders.

Purpose To evaluate the main etiological factors of severe cerebral palsy and comorbid pathology in children with severe motor disorders.

Material and methods A retrospective observational study included 170 patients with severe forms of cerebral palsy, divided into two groups (85 subjects each) depending on motor disorders: GMFCS IV, GMFCS V. Perinatal risk factors for severe cerebral palsy were assessed, correlations between perinatal risk factors for expressed movement disorders and height/weight indicators, comorbid pathology in children at the time of admission to the trauma and orthopaedic hospital.

Results Prenatal risk factors were responsible for the development of cerebral palsy in children in 71 % of cases. In the GMFCS IV group, gestational age had an inverse correlation with preterm birth ($R = -0.53$; $R^2 = 28\%$). In the GMFCS V group, disorders caused by a shorter gestational age were interrelated with the duration of the antenatal period ($R = -0.79$; $R^2 = 62\%$), and also directly correlated with delivery by cesarean section ($R = 0.58$; $R^2 = 34\%$). Among the comorbid pathologies, eye diseases and psychological development disorders were most often detected.

Discussion Low height/weight parameters of patients were due to comorbid pathology, rather than phenotypic constitutional features. Inverse correlation between the disorders caused by the gestational age, low birth weight and duration of pregnancy, risk of developing respiratory disorders, and a direct correlation with cesarean section seem logical. Severe comorbid diseases were more frequently diagnosed in patients with GMFCS V, indicating more extensive perinatal catastrophes in the central nervous system and the relationship between the developed pathology and severe motor disorders.

Conclusions The main risk factors for the development of cerebral palsy in patients with severe GMFCS IV–V motor impairments are associated with the pre- and intranatal periods. Comorbid pathology of patients with severe forms of cerebral palsy is caused by severe brain damage and movement disorders that have developed against this background.

Keywords: etiological factors, comorbid pathology, children, cerebral palsy

For citation: Evreinov VV, Zhirova TA, Zueva YaV. Main etiological factors and comorbid pathology in severe cerebral palsy. *Genij Ortopedii*. 2024;30(5):636–643. doi: 10.18019/1028-4427-2024-30-5-636-643

INTRODUCTION

The most common etiological factors contributing to the development of severe spastic forms of cerebral palsy (CP) are asphyxia, hemorrhagic or ischemic stroke, infections, central nervous system (CNS) malformations, and birth trauma [1, 2]. Premature birth (23–27 weeks of gestation) and low birth weight (less than 2500 g) are the most important predictors of CP, combined with persistent brain defects (cystic or cavum-like), severe movement disorders, dysphagia, neonatal seizures, and respiratory failure [2–4]. Maternal diseases (21–30.5 %) before conception (systemic diseases, use of psychotropic drugs, etc.) and during pregnancy (transient hypothyroxinemia, gestational diabetes, uterine bleeding, preeclampsia, etc.) also increase the risk of developing cerebral palsy in the child [5, 6].

Low height/weight parameters of patients with severe motor disorders due to cerebral palsy, detected at birth, persist with age, become most noticeable in adolescents and can reach z-scores lower than –2 [2]. In addition, such children are more often diagnosed with epilepsy (35–62 %), mental retardation (40–70 %), mental disorders (more than 50 %), dysarthria (40 %), visual impairment (from 40 to 75 %), hearing loss (4–13 %), oropharyngeal dysphagia (40–90 %), malnutrition (60–90 %), diseases of the genitourinary system (up to 60 %), chronic pain syndrome (32–74 %) [7, 8].

Thus, understanding the etiological causes of cerebral palsy allows for pre-pregnancy preparation of women for pregnancy, potential primary prevention of neurological disorders in newborns, while the understanding of probable comorbidities enables to plan early therapy for children with cerebral palsy, determine a rehabilitation strategy and a plan for social integration of such patients.

Purpose To evaluate the main etiological factors of severe cerebral palsy and comorbid pathology in children with severe motor disorders.

MATERIAL AND METHODS

A retrospective observational study included 170 patients (60 girls and 110 boys) with severe cerebral palsy, spastic dislocations (subluxations) of the femurs, in which reconstructive or palliative interventions on the hip joints were performed.

The work was carried out at the Ilizarov National Medical Research Center of Traumatology and Orthopaedics in the period from October 2021 to January 2024.

Inclusion criteria were:

- Age range: 5–17 years old;
- Severe CP (GMFCS IV–V);
- Uni- or bilateral spastic dislocation (subluxation) of the femur;
- Reconstructive or palliative interventions on the hip joint.

The patients were divided into two groups of 85 subjects each (30 girls and 55 boys) in accordance with the Gross Motor Function Classification System (GMFCS) of movement disorders. Children with severe motor impairments, unable to control body position and move without the help of parents (guardians), were assigned to functional level V (GMFCS V group), while patients who used technical rehabilitation devices for movement and could sit independently in a wheelchair were assigned to level IV (GMFCS IV group) [9].

Age and height/weight characteristics of the patient groups are presented in the Table 1.

Table 1

Age and height/weight parameters of patients in the groups, Me [Q1; Q3]

Parameter		GMFCS IV group	GMFCS V group	P
Age	Gestational age, months	31 [28; 32]	31 [28; 36]	0.06
	At time of surgical intervention, years	9 [6; 11]	9 [7; 11]	0.47
Weight, kg	At birth	1.5 [1.3; 1.8]	1.7 [1.3; 2.7]	0.04
	At admission to hospital	22 [17; 29]	18 [15; 23]	0.001
Height, cm	At birth	41 [39; 43]	41 [36; 48]	0.13
	At admission to hospital	*122 (16)	*120 (16)	0.34
Quetelet Index, kg/m ²	At birth	9.4 [8.4; 10.8]	10.1 [9.1; 11.6]	0.03
	At admission to hospital	15.5 [13.9; 17.7]	13.3 [12.4; 15.1]	< 0.001

Notes: Me — median; [Q1; Q3] — interquartile range; * — mean values and standard deviation (SD)

In the GMFCS IV group, spastic diplegia (Little's disease) was detected in 57 (67 %) patients, spastic tetraplegia in 28 (33 %) patients, while in the GMFCS V group it was 16 (19 %) and 69 (81 %) patients, respectively ($p < 0.001$) [10].

The severity of dysphagia was assessed according to the EDACS (Eating and Drinking Ability Classification System), which determines the ability to take food and liquid in everyday life [11]. In the GMFCS IV group, 24 (28 %) children were classified as level I, 36 (42 %) as level II, 25 (29 %) as level III, 2 (2 %) as level IV, while in the GMFCS V group: 12 (14 %), 19 (22 %), 38 (45 %), 16 (19 %), respectively, and were significantly different ($p < 0.001$).

Evaluation criteria:

- Perinatal risk factors for severe cerebral palsy;
- Correlations between perinatal risk factors for cerebral palsy, severe motor disorders according to GMFCS, and children's height and weight indicators;
- Comorbid pathology at the time of admission to the trauma and orthopaedic hospital.

Statistical processing of the material was performed using the Stat Plus 7 program. When the numerical values were subject to the Gaussian distribution criteria (Kolmogorov – Smirnov / Lilliefors), quantitative characteristics were described using the mean and standard deviation (SD). In cases where the assessed indicators did not meet the parameters of normal distribution, the median (Me) and interquartile range [Q1; Q3] were calculated. One-way analysis of variance or the nonparametric Mann – Whitney U-test were used to compare the groups. The χ^2 criterion was used to compare proportions. In all cases, the significance level α , at which the null hypothesis was rejected, was taken to be 0.05. The presence of a relationship between variables was determined by the pairwise linear correlation coefficient (R), and the strength of the relationship was determined by the Chaddock scale. The proportion of variance was estimated by the determination coefficient (R²). The study was approved by the institutional ethics committee (protocol No. 2 (70) dated October 21, 2021) and was conducted in accordance with the ethical standards set out in the Declaration of Helsinki.

RESULTS

Prenatal risk factors were responsible for the development of cerebral palsy in 71 % of cases, while intranatal factors were responsible for 21 % and postnatal factors were responsible for 8 % of cases. The main predictors of intrauterine cerebral palsy included surgical or spontaneous abortions in the mother's history, bleeding and anemia during pregnancy, gestosis, sexually transmitted infections (STIs), pelvic anomalies, and fetal pathologies. The most significant etiologic factors during labor were short gestational age and birth weight, cesarean section, and respiratory and cardiovascular disorders in the newborn. Factors that could lead to brain damage of the newborn in the extrauterine period were sepsis and hemolytic disease (Table 2).

Table 2

Perinatal risk factors for CP in the groups

Risk factors for CP (ICD10)	Group				P
	GMFCS IV, n = 85		GMFCS V, n = 85		
	Number	%	Number	%	
Prenatal					
Coagulation disorders (D68)	3	3.5	0		0.08
History of pregnancy with an abortive outcome (O00–O08)	41	48.2	36	42.3	0.44
Edema, proteinuria and hypertensive disorders during pregnancy (O10–O16)	35	41.2	45	52.9	0.12
Bleeding in early pregnancy (O20)	49	57.6	45	52.9	0.53
Urinary tract infection during pregnancy (O23)	9	10.5	12	14.1	0.48
Multiple pregnancy (O30)	14	16.5	5	5.9	0.02
Malpresentation of the fetus (O32)	1	1.2	6	7	0.05
Established or suspected maternal pelvic anomaly (O34)	14	16.5	11	12.9	0.51
Established or suspected pathological conditions of the fetus (O36)	14	16.5	10	11.7	0.37
Polyhydramnios (O40)	6	7	0		0.01
Disorders of the amniotic fluid and fetal membranes (O41)	6	7	6	7	1
Placenta previa (O44)	2	2.4	6	7	0.14
Premature placental abruption (O45)	17	20	15	17.6	0.69
Infectious and parasitic diseases of the mother complicating pregnancy (O98)	11	12.9	14	16.5	0.51
Anemia complicating pregnancy (O99.0)	16	18.8	19	22.3	0.57
Diseases of the endocrine system, nutritional disorders and metabolic disorders that complicate pregnancy (O99.2)	3	3.5	7	8.2	0.19
Intrauterine hypoxia (P20)	13	15.3	6	7	0.08
Respiratory diseases complicating pregnancy (O99.5)	17	20	8	9.4	0.05
Restoration and preservation of reproductive function (Z31)	8	9.4	1	1.2	0.01
Problems related to the mother's lifestyle (Z72)	1	1.2	2	2.4	0.56
Intranatal					
Premature rupture of membranes, onset of labor after 24 hours of anhydrous period (O42.1)	13	15.3	11	12.9	0.66
Preterm labor and delivery (O60)	81	95.3	68	80	0.00
Delivery by caesarean section, with forceps or with the use of a vacuum extractor (O81–O82)	64	75.3	66	77.6	0.71
Disorders associated with shortened gestation and low birth weight (P07)	80	94.1	64	75.3	< 0.001
Respiratory and cardiovascular disorders characteristic of the perinatal period (P21–P29)	77	90.6	82	96.5	0.12
Intracranial nontraumatic hemorrhage in the fetus and newborn (P52)	24	28.2	19	22.3	0.37
Postnatal					
Bacterial sepsis of the newborn (P36)	7	8.2	6	7	0.77
Hemolytic disease of the fetus and newborn (P55)	1	1.2	1	1.2	1

Based on the correlation analysis, a moderate inverse relationship was found between the severity of motor disorders according to GMFCS and the body mass index at the time of admission to the trauma and orthopaedic hospital ($R = -0.32$). The coefficient of determination (R^2) of the dependent variable

(BMI) was 10 %. The strength of the relationship according to Chaddock among perinatal risk factors for cerebral palsy and major motor disorders was either absent or very weak. Also, no relationship was registered in both groups between the Quetelet index (BMI) at birth and the BMI upon admission to the Ilizarov Center.

In the GMFCS IV group, the gestational age was directly correlated ($R = 0.59$; $R^2 = 35\%$) with the birth weight index and had an inverse correlation with preterm birth ($R = -0.53$; $R^2 = 28\%$), the risk of developing respiratory and cardiovascular disorders in the neonatal period ($R = -0.43$; $R^2 = 18\%$) (Table 3). In the GMFCS V group, disorders due to shortened gestational age and low birth weight were interconnected with the duration of the antenatal period ($R = -0.79$; $R^2 = 62\%$), and also directly correlated with cesarean section ($R = 0.58$; $R^2 = 34\%$) (Table 4).

Table 3

Correlation coefficients of parameters in group GMFCS IV, $n = 85$

Parameters	O60	P07	P21–P29	Gestational age, months	BMI at birth, kg/m ²
Preterm labor and delivery (O60)	1				
Disorders related to short gestation and low birth weight (P07)	0.43	1			
Respiratory and cardiovascular disorders specific to the perinatal period (P21–P29)	0.24	0.23	1		
Gestational age, months	–0.53	–0.35	–0.43	1	
BMI at birth, kg/m ²	–0.52	–0.51	–0.19	0.59	1

Table 4

Correlation coefficients of parameters in group GMFCS V, $n = 85$

Parameters	O32	O36	O40	O60	O81–82	P07	P20	Gestational age, weeks	BMI at birth, kg/m ²
Malpresentation of the fetus (O32)	1								
Established or suspected pathological conditions of the fetus (O36)	–0.03	1							
Polyhydramnios (O40)	–0.05	–0.02	1						
Preterm labor and delivery (O60)	0.03	–0.08	0.50	1					
Delivery by caesarean section, with forceps or with vacuum extraction (O81–82)	0.13	0.06	–0.05	0.27	1				
Disorders related to short gestation and low birth weight (P07)	0.16	0.07	–0.16	0.15	0.58	1			
Intrauterine hypoxia (P20)	0.42	0.49	–0.04	–0.02	0.12	0.14	1		
Gestational age, months	–0.21	0.12	0.16	–0.20	–0.55	–0.79	–0.09	1	
BMI at birth, kg/m ²	–0.19	–0.05	–0.07	–0.10	–0.28	–0.29	–0.13	0.40	1

In the compared groups, among the comorbid pathologies, eye diseases and disorders of psychological development were identified; in patients with extremely severe motor disorders (GMFCS level V), diseases of the genitourinary, nervous (epilepsy, hydrocephalus, etc.), endocrine systems, nutritional disorders and metabolic disorders were most often diagnosed (Table 5).

Table 5

Comorbid pathology in the groups of patients

Коморбидная патология	Group				<i>P</i>
	GMFCS IV, <i>n</i> = 85		GMFCS V, <i>n</i> = 85		
	Abs. number	%	Abs. number	%	
Diseases of the eye and its adnexa	72	84.7	66	77.4	0.24
Diseases of the skin and subcutaneous tissue	1	1.2	1	1.2	1
Diseases of the blood, hematopoietic organs and certain disorders involving the immune mechanism	3	3.6	3	3.6	1
Diseases of the genitourinary system	26	30.6	56	65.8	< 0.001
Diseases of nervous system	38	44.7	52	61.2	0.03
Diseases of respiratory organs	10	11.7	10	11.7	1
Diseases of the digestive system	22	25.8	55	64.7	< 0.001
Diseases of blood circulation system	10	25.9	15	17.6	0.27
Diseases of the endocrine system, nutritional disorders and metabolic disorders	48	56.5	67	78.8	0.004
Congenital anomalies, deformities and chromosomal disorders	8	9.4	14	16.5	0.17
Disorders of psychological development	76	89.4	75	88.2	0.8

DISCUSSION

In our study, most of the risk factors associated with the likelihood of cerebral palsy in a child were related to the period of embryonic development that is consistent with large studies in this area [12–15]. Hypoxic-ischemic damage to neurons and intracranial hemorrhages caused the formation of epileptic foci in the brain, severe intellectual disorders, spastic diplegia and tetraplegia, pseudobulbar disorders, and oropharyngeal dysphagia. Along with this, low height/weight parameters of patients in the groups, diagnosed at birth and persisting with age, were probably due to comorbid pathology, severe motor limitations, low nutrient intake, energy deficiency, metabolic disorders, and not phenotypic constitutional features [16–21].

Premature birth is often promoted by acute inflammatory diseases of the female pelvic organs, a history of abortions, and a postoperative scar on the uterus [22, 23]. In such cases, obstetricians and gynecologists use surgical methods of delivery to reduce neonatal mortality [24]. In a newborn, due to morphologically immature lungs, gas exchange may be impaired, metabolic acidosis, and respiratory failure may develop, which may lead to hypoxic ischemia of the myocardium and brain of the child [25–27]. Thus, the inverse correlation revealed in our work between disorders caused by the gestational age, low birth weight, and the duration of pregnancy, premature birth, the risk of respiratory and cardiovascular disorders and a direct correlation with cesarean section delivery seems logical.

According to the US Centers for Disease Control, the mortality rate of infants born at 22–24 weeks of gestation is 64 %, and neurological disorders are detected in 43 % of cases among survivors [25, 28]. Extremely premature infants have white matter lesions in the form of periventricular leukomalacia or consequences of hemorrhages in 80 % of cases, while full-term infants have a gray matter defect [12, 15]. Thus, bilateral spastic forms of cerebral palsy were recorded in 31.4 % of cases, mental retardation with IQ < 50 in 32.1 % of cases, and severe eye vision impairment in 12.3 % of cases [15, 29, 30]. In our study, cerebral vision impairment and retinopathy of premature children were the main eye diseases in children with severe limitations of motor functions [31]. Psychological disorders were also established with equal frequency, which indicates damage to similar parts of the brain. At the same time, diseases of the genitourinary, digestive, endocrine systems, nutritional

disorders and metabolic disorders, hydrocephalus, epilepsy were more often diagnosed in patients with GMFCS V, which indicates more large-scale perinatal catastrophes that occurred in the central nervous system, as well as the relations between the pathology developed and pronounced motor disorders.

CONCLUSION

The main risk factors for the development of cerebral palsy in patients with severe motor impairments GMFCS IV–V are associated with the pre- and intranatal periods.

Comorbid pathology in patients with severe cerebral palsy is caused by severe brain damage and motor disorders that have developed due to it.

Conflict of interest The authors declare no conflicts of interest related to the publication of this study.

Funding Not declared.

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The article was submitted 14.03.2024; approved after reviewing 21.03.2024; accepted for publication 01.08.2024.

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